ANGINA AND OTHER PRECORDIAL PAIN—THEIR DIFFERENTIATION*

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ANGINA is an old and favorite subject for medical discussion, and there seems to be little opportunity to throw new light on a topic which has received so much attention from the masters of medicine since the time of Heberden. And yet the "physiognomy" of angina is still often hazy and surrounded by confusing shadows; and it is well that we give it from time to time a careful scrutiny.

WHAT IS ANGINA?

What is angina, what is mock angina, what is the mechanism of the pain and of death, and what is the pathology? The answers are still colored heavily with speculation and opinion; and an impressive testimony to the unsettled state of opinion was furnished by Huchard when he enumerated sixty-four theories as to the cause of angina. According to fashion, the theories have grouped about questions of heart spasm, arterial pressures, coronary spasm or sclerosis, esophageal traction, etc. Allbut argued fervently for his theory of tension within a sensitive aorta, and Mackenzie for his concept of angina as a symptom of myocardial exhaustion, comparable to the leg pain of intermittent claudication or the pain from any muscle if worked out of proportion to its blood supply. The known facts are that most cases have some cardiovascular pathology, commonly aortic, coronary or myocardial lesions, but that these are at times slight and occasionally absent in fatal cases. And the only mechanistic conception adequately covering these facts is that taught for many years by Bamberger and Von Neusser, namely, that of an excitation in the vegetative nervous system, which may arise from various sources in and about the heart, and resulting afferently in the sensation of pain or constriction, and liable efferently, through the sensorimotor reflex arc, to stop the heart. The latter effect is usually assumed to be a simple vagal standstill; but, coming as it usually does without preliminary slowing, it is better accounted for by ventricular fibrillation.

At any rate, such a concept is a convenient frame on which to arrange our ideas of diagnosis. One case we may feel is aortitis with an enlarged aorta and no evidence of myocardial trouble, another appears to be myocardial, or again, we may feel sure of a coronary thrombosis; and with all we sense the possibility of death through the sensorimotor reflex. Now by the term "angina" we refer to all cases and only those in which such a death is considered possible; and our serious practical problem is to separate these from other confusing types of pain which convey no such threat.

CONDITIONS WHICH SIMULATE ANGINA

One need only mention the necessity of ruling out such conditions as erosive aneurysm, ordinary pleurisy and pericarditis, tabetic pains and herpes. Nerve-root pains, so carefully described by Gunther, especially in spinal arthritis, are distinguished by their sharp delimitation along the distributions of the affected nerves and by their excitation by motions of the spine. Many of these are called intercostal neuralgia or neuritis.

Then there is a miscellaneous assortment of precordial pains variously designated as nervous pain, fatigue pain, toxic or secondary angina, or pseudo-angina. Much as we desire an etiological classification, we had better honestly admit that we can give no convincing explanation for many of these pains; and to my mind the much condemned term "pseudo-angina" serves the useful purpose of designating them without pretending to understand them, and it emphasizes the all-important distinction from true angina.

DIFFERENTIAL DIAGNOSIS

To proceed at once to the differential diagnosis, it may be said that laboratory procedures, and even the physical examination, is of minor utility when compared with history, for the thing we are differentiating is a symptom complex. The x-ray and electrocardiograph are often useful in corroborating clinical impressions, and at times supply practically our only positive evidence of aortic or myocardial disease. A positive Wassermann test may help in a doubtful case. The general type of the patient helps somewhat. Males in middle age or beyond, in high pressure business or professional careers are candidates for angina. Younger subjects or women at the menopause, and neurotic types generally are more likely to have pseudo-angina. Abnormal blood pressure and cardiac murmurs, even those significant of valve damage, help but little except insofar as they contribute to our conception of the state of the aorta, the coronaries and the myocardium; and it must be remembered that such circulatory abnormalities do not protect against false angina. Poor heart sounds, some of the gallop rhythms and graver types of disordered heart-beat mechanism may, of course, point definitely to myocardial trouble; and pericardial friction, in association with other suggestive evidence (pain, prostration, poor pulse, fever, leukocytosis), may, of course, make certain the diagnosis of coronary thrombosis.

CLINICAL HISTORY

The *history* begins with the family. A minute will suffice to determine whether or not a patient has any information of value about his blood relatives. If he has, then our perfunctory formulae for recording family history should be ampli-

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fied to include all the data available. Certain families are so consistently of the cardiovascular type or of the nervous type that one almost knows what he is to find in the patient. The past history of the patient's life may be of some help if it suggests, on the one hand, rheumatic, streptococcic or syphilitic infection, or on the other neurotic tendencies.

Habits of eating, drinking and smoking are often excessive in the angina patient and abstemious in the neurotic type. Tobacco undoubtedly is at times a cause, at least an exciting cause, of angina, as in the case reported by Lambert of a man who lived comfortably three months without tobacco and then dropped dead after smoking a part of one cigar. Such cases are spectacular but, fortunately, rare, and they have served to overemphasize in the mind of the profession the importance of tobacco as an etiological agent. In this connection it is interesting to recall the experience of the British heart hospital during the war. Most of their cases had pain, weakness, and various other functional disturbances—the sort often loosely referred to as the "cigarette heart." But it was found that, whereas they were culled from an army of almost universally heavy smokers, these patients for the most part smoked little or not at all. The inference, of course, is not that tobacco protects the heart, but simply that the kind of man who develops the effort syndrome or pseudo-angina is also the one who is likely to eschew excesses.

STUDY OF SYMPTOMS

In the present illness the pain should be analyzed systematically under four principal headings. In the order of importance they are:

1. *The Kind of Pain.*—Two types stand out clearly. One may be called *compression pain.* Patients who have it will usually volunteer the description in such terms as "vise-like," "terrible oppression in the chest," "heavy weight behind the breast bone," "petrified feeling" (pointing to central part of chest), "belt being cinched about the windpipe," "pent-up gas behind the breast bone," etc. The onset may be described as a sudden seizure, but as a rule careful inquiry will bring out the observation of a shorter or longer period of crescendo, and also a decrescendo as the peak is passed and the anguish melts away. Of these the vast majority are instances of true angina.

The other distinctive kind of pain may be termed *lancinating pain.* There is no crescendo here, maximum intensity being experienced on the instant. When severe it is like a "stroke of lightning," a "knife" or a "red-hot needle" plunged through the heart. One young man was felled instantly to the floor by it while quietly working in a power station; another while strolling about the hospital where he was convalescing from a respiratory infection; another young man was instantly "crumpled up into a helpless mass" by it while driving an automobile. These major instances of lancinating precordial pain are uncommon; but from them we can descend the scale of intensity through various "stabbing," "jabbing," "grabbing" sensations, etc., until we arrive at the trifling "nip" or "stitch in the side" at some time or other experienced by most people. And it is this gradation without variation except in intensity that stamps typical lancinating precordial pain as pseudo-angina. When unassociated with compression sensation, I have never known a case of lancinating pain to result fatally. Seven years ago a doctor, sixty-five years old, experienced without prodromata and without apparent provocation a terrific instantaneous knife-like pain through the precordium and left arm which lasted all night and alarmed me at the time. But he is still in active work and has had no recurrence of pain and has normal physical findings and electrocardiogram. Typically, lancinating pain lasts but a moment, though it is often succeeded, if severe, by a dull ache. While it lasts the subject usually holds the breath, feeling, as one expressed himself, that "If I breathed it would tear me in two." Ordinarily far apart, the paroxysms may come in groups. One of my patients and one reported by Thomas Lewis described feeling something "like an electric shock" with each heart beat for hours at a time, starting from the region of the cardiac apex and instantly passing up and into the left arm and out the finger tips.

After these two distinctive types of pain comes a miscellaneous group of dull aches, sore, or burning feelings, etc., which may alternate with the compression pain of angina, but are more common in pseudo-angina. Terrific or simply "awful" indescribable pain in the chest is always threatening. In coronary thrombosis it is perhaps even more common than typical compression pain.

2. *Factors which precipitate or relieve attacks* come next in importance in differential diagnosis. The pain of coronary blockage, though often appearing in sleep or after a full meal, as a rule has no recognizable precipitating cause. It is comparatively of long duration, is little if at all affected by nitrites and often requires large doses of morphin for relief. With this exception, true angina pain is practically always precipitated by some well-defined circumstance or train of circumstances. Here, however, the uniformity ends. Almost every patient has his idiosyncrasies. The classical precipitant is walking up hill against the wind after a meal. Some go about freely on a warm quiet day, but cannot stand facing a cool breeze without having pain. One of my patients with syphilitic aortitis and valvulitis who could chop wood at mid-day could not so much as step out of his door in the morning or evening. A drink of cold water would give him an attack. Another with the same syphilitic lesion would be held up by pain while sauntering on a warm day to the beach, and would then enter the cold water and swim vigorously with no discomfort whatsoever. Another man with nonsyphilitic aortitis cannot take a hot drink. He is a retired engineer,

and it is his hobby to prepare graphs of his pain threshold for varying temperature of drinks at different times of day. Some have attacks from emotion rather than physical exertion; some from use of certain muscles, especially the left arm; some merely from palpating the precordium. The physiological alterations in sleep are little understood but are often important in angina, and the waking hour is a critical one for many patients. Nocturnal attacks are usually of ill omen.

Now from angina patients these precipitating factors as a rule are elicited with little or no direct questioning. Not so with pseudo-angina patients. They will often deny any association of pain with meals, exercise, excitement, etc.; or if such a relationship is claimed it will probably be found to be a delayed one. After a hard day they may have pain while quietly resting that evening or the next day. Pseudo-angina may, however, follow close on the heels of a family row for example.

Relief of pain following discontinuance of the exciting cause is a valuable point in favor of angina. Vasodilators as a rule relieve angina, except in coronary thrombosis. They have much less effect on pseudo-angina. Lancinating pain rarely lasts long enough for any remedy to be tried.

3. *Topography of the pain* is less valuable in differentiation than is often supposed. Contrary to a widespread preconception true angina is much more commonly, and as originally described by Heberden, substernal; while false angina is usually precordial. True anginal pain has usually a very blurred delimiting outline in the chest, the patient often indicating location vaguely by a wave of the hand; though when it passes on to the arm, neck, etc., the pain outlines are often sharp. Pseudo-angina dull pain may be hazy in outline, but is more often fairly sharp. Lancinating pain, in particular, is usually indicated by the patient as a definite point or streak, which, however, may vary in different attacks (sometimes the same thing occurs in the right chest and occasionally in remote parts of the body). It is interesting that when patients describe such a streak of pain it is not along the course of any nerve, but is from the central or lower precordia upward toward the shoulder, straight through to the back, or diagonally up and across the sternum, etc.

In general, pseudo-anginal pain radiates less commonly than anginal, but not infrequently it extends into the typical left arm distribution. Radiation in angina is usual and is quite bizarre. Instead of the left ulnar distribution it may go only to the right arm, or to both arms, the back, neck, jaw, or occiput. Osler described a radiation to the left testis. One must at times recognize angina with pain only at the area of pain reference. One of my patients who had syphilitic aorta and heart had only stinging effort pain in both wrists; another, a doctor, began having his teeth pulled because of effort pain limited to the left jaw but which later began in the chest and radiated to the jaw.

Abdominal angina is not to be forgotten. Deaths described in the newspapers as caused by "ptomaine poisoning" or "acute indigestion" are usually of this sort. Especially to be remembered is the occasional case of coronary thrombosis with epigastric pain, tenderness and rigidity, together with fever and leukocytosis. Pericardial friction may establish the diagnosis, or it may be determined by careful observation of the pulse and electrocardiogram.

4. The *psychic state and various other associated phenomena* of the attacks are to be considered. Pallor and immobility during attacks and salivation and polyuria afterward are more characteristic of angina. Flushing of the face and a demonstrative behavior are more like false angina. In the angina attack there may be a sense of suffocation, with natural or suppressed breathing, or the patient may expand the chest in the effort to get relief. In pseudo-angina, especially with lancinating pain, the breathing is more often held within shallow limits for fear of increasing the pain. In either condition the heart rate may be retarded or accelerated, the pulse strong or weak, the blood pressure higher, lower or unchanged.

Skin tenderness for hours or days is common after a severe attack of angina; it may be acute over the precordium or the area of pain reference or both. It is also common in cases of pseudo-angina, but here is more often limited to the region of the heart apex, and is rather less closely connected with the attacks. Many pseudo-angina patients describe habitual sensitiveness below the left nipple, which may not be so much actual tenderness as vague discomfort produced by even the contact of clothing. Aversion to lying on the left side (occasionally it is the right side) on account of pain, heart consciousness, smothering sensations, etc., is much more characteristic of the nervous group.

The textbook "angor animi," the paralyzing sense of impending dissolution, when it occurs, is a warning that no physician will fail to heed. But if he waits for this signal to establish the diagnosis he will miss most of the cases. On the other hand, many of the most badly frightened people I have seen were clearly suffering pseudo-angina; and, whereas typically their state of mind is supposed to be a fear that they may have angina rather than the simple conviction of doom, in practice it is often hard to draw so sharp a distinction. I would go so far as to characterize reliance on this fear symptom as positively dangerous. The principal object of diagnosis is therapy; and there are few instances where failure of diagnosis may more seriously jeopardize the success of treatment. For when the physician, overimpressed by the fear of the sufferer from pseudo-angina, takes the view that it is or that

it may be the real thing, he will almost certainly fail to administer the all-important convincing reassurance, and the chains of neurosis will be welded.

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DISCUSSION

WILLIAM DOCK, M. D. (Stanford University Hospital, San Francisco).—This journal has recently had an unusually good series of papers on angina pectoris, including those of Doctors J. M. Read and W. S. Thayer. Doctor Kilgore's paper rounds out this series very well, and, so far as his conclusions upon diagnosis and treatment are concerned, I have nothing to add. There is apparent throughout this recent series considerable difference of opinion as to terms, but agreement as to facts. I should differ with Doctor Kilgore on terms. I prefer to think of angina pectoris as that type of heart distress, never associated with dyspnea of the panting type which was described by Heberden. I, therefore, do not make the diagnosis if some other cause for the precordial pain is evident—that is, if the patient has coronary occlusion, paroxysmal tachycardia, or merely palpitation and "heart consciousness." Pseudo-angina seems to me as poor a term as "pseudo-epilepsy" or "pseudo-tuberculosis." In atypical cases there will be greater uniformity of diagnosis of angina pectoris by experienced physicians than there will be in cases of pulmonary tuberculosis. The practice of lumping all precordial pain into the groups "angina" and "pseudo-angina," though supported by men as expert as Sir James Mackenzie and Doctor Kilgore, leaves much to be desired.

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T. HOMER COFFEN, M. D. (707 Stevens Building, Portland, Oregon).—Since Osler's Lumleian lectures on angina in 1910, Herrick's paper on coronary thrombosis in 1911 and Allbut's discussion of anginal pain in 1915, medical literature has shown increasing volume on this subject. Naturally, in differentiating angina from other conditions which simulate it, there has been much discussion, and certain underlying causes stand out as definite entities, such as coronary thrombosis. Some cases of precordial pain lead one to a certain feeling of assurance in assuming that coronary sclerosis is present, especially if peripheral arteries are thickened and if the aorta is increased in density or widened under the fluoroscope. In fact there has been a distinct tendency in the last few years to consider angina synonymous with underlying disease of the coronary arteries, though Dr. Thomas McRae's recent paper sounded the warning that certain cases with anginal symptoms have no coronary disease. One must not overlook the fact that some cases of real angina have masked symptoms. For instance, not a few of my patients have had symptoms entirely referred to the epigastrium and lower sternum. The recent work of Whitten and Barnes seems to show that further correlation of clinical knowledge with that obtained at the autopsy table may enable us to recognize right and left coronary occlusion. I suggest that we consider angina or the anginal syndrome, a general term of which coronary thrombosis, coronary sclerosis or spasm are varieties.

It is certainly true that if we review the histories of a few hundred people who have consulted us regarding precordial pain we find between 30 to 40 per cent in whom we eliminate angina as a causative factor; they fall in the group to which Doctor Kilgore refers as false angina. The etiology of these cases has been well discussed by him. In diagnosis I agree with him that the history is important. A therapeutic test which I occasionally use is nitroglycerin for the attack. That certain people are sensitive to tobacco, "tobacco angina," has been demonstrated by instances in my own experience of men, usually young, who have found that tobacco precipitates precordial pain. Physical and laboratory examinations show no abnormalities in these patients.

DONALD J. FRICK, M. D. (1136 West Sixth Street, Los Angeles).—Doctor Kilgore, in his paper on "Angina and Other Precordial Pain," tersely covered his subject in a most satisfying manner. What I have to say is at the same time irrelevant and pertinent.

Many of us have long felt the undesirability of the term "angina pectoris." It has formed a diagnostic scrap-basket; one of those generic names which suggests so much, yet specifies so little. It would be as appropriate to use the term "angina abdominis" for reflex pain due to renal colic, mesenteric thrombosis, or visceral perforation.

Of late years we have found hidden under the inclusive term (angina pectoris) a number of distinct pathologic conditions involving the heart, the great vessels and other thoracic organs. The retention of this title hinders the thorough sifting and classification of these various conditions. Accepted as a cause of death, it nullifies vital statistics in heart disease.

If this term could be discarded in our generation, what a boon it would be to the laity of the future. Without this diagnostic smoke screen, search for the cause of chest pain would be more thorough and treatment more intelligent. Then, too, the victim of precordial pain might be spared the casual diagnosis and the persistent fear that follows in its wake. A fear reborn with every news item of sudden death from angina pectoris. A fear that is present from the first mention of the term to the end of the patient's life.

The patient could be told he had intercostal neuritis, if that were true, and the heart be left out of the discussion. In another instance, coronary thrombosis could be explained and the wisdom of rest and future caution in activity pointed out. In neither case would this much dreaded term be mentioned, and the patient would live out his life in reasonable peace of mind.

It is, then, for the sake of the patient and the physician, with peace for the former and scientific advancement for the latter, that angina pectoris should be thrown in the discard.

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DOCTOR KILGORE (Closing).—In view of most modern utterances on the terminology, I was expecting the attack on pseudo-angina, but not Doctor Frick's repudiation of even the time-honored angina pectoris. Certainly both terms should be replaced by names designating specific morphologic changes or functional mechanisms deviating from normal, *if we could do so confidently in all, or even in most cases.* But I cannot do that, and so I do not share Doctor Frick's optimism that the average doctor can do it and that he would better our vital statistics by trying to do it. It is as often the exception as the rule that I can predict with assurance and with success the necropsy findings in undoubted cases of angina; and far less can I give a convincing explanation morphologically or functionally of the cases to which I refer as pseudo-angina. They are not tabes, pleurisy, herpes, and the like. And the use of such terms as "intercostal neuritis" is a gesture of knowledge with a conviction of ignorance. The name "pseudo-angina" frankly connotes our ignorance of mechanism, while it affirms the all-important distinction from angina, and, therefore, when understood should give the maximum protection against anxiety neurosis. Yes, one could even approve of Doctor Dock's "pseudo-tuberculosis" if the situation were in the least degree comparable, i. e., if tuberculosis were a symptom complex instead of a specific infection, and if its imitators were so ill identified and appeared under so many aliases as those of angina. However, we should all agree that the terminology is of secondary importance. The real task is the diagnostic one of separating the pain with a threat from the one without it, and in this task in actual practice there is happily less disagreement.